stage larvae within their natural intermediate hosts, such as slugs and snails, notably the *Pila* species commonly eaten in Thailand. Freshwater prawns, land crabs, and frogs have been found to harbor the third-stage larvae of the parasite as a result of eating infected intermediate hosts. Humans, like rodents, become parasitized when they ingest infected molluscs, contaminated vegetables, or fomites. Larvae migrate to the brain, where they grow and subsequently travel to the lungs to deposit eggs. In humans, the nematode does not complete its life cycle, usually dying after reaching the central nervous system.[3]

A cantonensis infections are usually mild, with headache, nausea, photophobia, and neck stiffness as the predominant symptoms.[2] Severe peripheral paresthesia may be a diagnostic clue. Generalized weakness, visual disturbances, and extraocular muscle palsies are also well described,[2,3] but urinary retention, ataxia, and spinal cord lesions in humans are rare. Fever exceeding 38°C is uncommon.[5] Hepatic and pulmonary involvement have not been previously reported. Although not proved in the first case, no other cause was found for the pneumothorax and bronchopleural fistula or for the abnormal values on liver function tests. Resolution of the abnormal liver function correlated with the clinical resolution of the illness and with the administration of steroids.

Most cases of angiostrongyliasis in humans are self-limited, and recovery without sequelae is the rule. A number of cases of greater severity and chronicity have been reported, however, and these may correlate with a higher parasite load.[1,6] In patients 2, 3, and 4, the severity of the illness was directly related to the amount of unwashed lettuce each patient consumed.

The diagnosis of *A cantonensis* meningitis is suggested by the triad of typical clinical presentation, a history of exposure through eating intermediate hosts (such as shellfish) or contaminated food (such as unwashed watercress or lettuce), with cerebrospinal fluid eosinophilia. Staining of CSF for eosinophilia must usually be specifically requested. Positive serologic testing using an enzyme-linked immunosorbent assay is confirmatory.[7]

The differential diagnosis of *A cantonensis* meningitis includes cerebral cysticercosis, the only other infection in the Pacific region documented to occasionally cause CSF eosinophilia in excess of 10%.[8] Cerebral involvement with this parasite is rare and will usually present with symptoms or signs of a space-occupying lesion. In Southeast Asia, particularly Thailand, an intense eosinophilic pleocytosis can also be caused by *Gnathostoma spinigerum*.[3] The presenting symptom is often sharp pain from irritation of a nerve root. Myeloencephalitis with limb paralysis is common, and severe disabling sequelae may occur. The CSF is often bloody or xanthochromic. A serologic test for *Angiostrongylus* species does not cross-react with that for *G spinigerum*. *G spinigerum* is not known to occur in Australia or the Pacific Islands.

There have been a few reports of CSF eosinophilia associated with *Toxocara canis, Trichinella spiralis, Ascaris lumbricoides, Echinococcus granulosus,* and *Strongyloides stercoralis* infections.[8] In these cases, CSF eosinophilia is usually less than 10% and may be purely a result of intense peripheral eosinophilia. Similarly, neurosyphilis, tuberculous meningitis, and lymphomas may uncommonly cause mild CSF eosinophilia, but not to the degree seen in our patients.[8]

The optimal regimen for treatment is not established. Anecdotal reports exist for the efficacy of thiabendazole[3,9] and diethylcarbamazine citrate,[10] but anthelmintic drugs have not been shown conclusively to be beneficial in humans. Corticosteroids may be helpful in alleviating symptoms of raised intracranial pressure and in reducing allergic reactions to living or dead larvae.[2]

The disease is largely prevented through the control of rats. The proper washing of vegetables and cooking of snails and aquatic crustaceans are also important.

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Accidental Poisoning by Warfarin-Contaminated Herbal Tea

WILLIAM A. NORCROSS, MD
THEODORE G. GANIATS, MD
LEE P. RALPH, MD
RAMONA G. SEIDEL, MD
TYSON S. IKEDA, MD
La Jolla, California

AMERICAN CITIZENS, especially those living near the United States-Mexico border, frequently shop in Mexico. We report the case of a patient presenting with a coagu-

(Norcross WA, Ganiats TG, Ralph LP, Seidel RG, Ikeda TS: Accidental poisoning by warfarin-contaminated herbal tea. West J Med 1993; 159:80-82)

From the Department of Family and Community Medicine, University of California, San Diego, School of Medicine, La Jolla.

Reprint requests to William A. Norcross, MD, Dept of Family and Community Medicine, UCSD Medical Center, 225 Dickinson St, San Diego, CA 92103-8809.

lation disorder apparently engendered by drinking warfarin-contaminated hibiscus tea purchased in Tijuana.

Report of a Case

The patient, a 41-year-old woman with a history of interstitial cystitis, was seen because of hematuria and left flank pain for two days. A urinalysis revealed yellow color, slight proteinuria (1+), and an erythrocyte count of more than 50 per high-power field. A urine specimen was submitted for culture, and a three-day course of the combination drug of trimethoprim and sulfamethoxazole was started. The patient was seen two days later with continued flank pain and no response to the antibiotic therapy. The urine culture showed no growth. An intravenous pyelogram done that day was normal. The patient was treated with analgesics, and on the following day she noted that the flank pain, though still present, was notably diminished. She also had a recent onset of mild headache, fatigue, and mild dyspnea on exertion, and over the past several months she had noticed easy bruising. A physical examination revealed no abnormalities other than several small ecchymoses distributed on the trunk and the lower extremities. A urinalysis showed rare leukocytes and erythrocytes on microscopic examination. On a complete blood count she had a hematocrit of 0.27 (27%) and a hemoglobin of 87 grams per liter (8.7 grams per dl) with normal indices. The leukocyte count and differential cell count were within normal limits. The platelet count was 534×10^9 per liter. The patient's prothrombin time was 30.0 seconds with a control of 12.3 seconds (international normalized ratio [INR] 11.5).* Her partial thromboplastin time was 36.8 seconds with a control of 27.9 seconds. The prothrombin time performed with a 1:1 mix corrected to 12.5 seconds with a control of 12.1 seconds (INR 1.1); the partial thromboplastin time with a 1:1 mix corrected to 28.1 seconds and a control of 27.2 seconds. Factor II and factor VII levels were 6% and 5% of normal, respectively.

The patient was admitted to the hospital with the diagnosis of coagulation disorder due to prothrombin-complex deficiency. An extensive history failed to disclose any possible sources of purposeful or accidental warfarin ingestion. Phytonadione, 10 mg, was administered subcutaneously daily and then iron sulfate, 325 mg, orally daily. Within 60 hours of admission, the patient's prothrombin time had dropped to 13.5 seconds with a control of 12.2 seconds (INR 1.3). Her symptoms resolved over the ensuing days, and her recovery was uneventful. A high-performance liquid chromatographic assay of her serum (Hewlett-Packard 1090 high-performance liquid chromatograph using UV detection at 280 nm, performed by Poisonlab, Inc, San Diego, California) returned positive for warfarin. At the time of discharge, extensive efforts by the patient and her physicians failed to elucidate a plausible source for warfarin ingestion. The patient was specifically asked about the possibility of a surreptitious administration of warfarin by herself or family members and denied any possibility of this.

During a follow-up visit five days after discharge, the patient told her family physician of her suspicions that one or more of several herbal teas that she had purchased in Tijuana might contain warfarin. The patient deduced this possibility because her husband and children were found to have normal prothrombin times, and this was the only substance ingested solely by her. Several samples of brewed tea were sent for warfarin assays. One hibiscus tea sample (Lagg's Jamaica Tea, manufactured in Mexico City) returned positive for metabolic products of racemic warfarin (same method and company). This was immediately reported to the San Diego County Department of Health Services, the Food and Drug Branch of the California Department of Health Services, the United States Food and Drug Administration, and the appropriate public health agencies in Mexico. In addition, the tea manufacturer was notified.

Discussion

The anticoagulant effect of bishydroxycoumarin was first reported in 1922 by Schofield, who described a hemorrhagic disease of cattle following feeding on spoiled sweet clover silage.[1] This came to be known as "sweet clover disease" and led to the discovery of the anticoagulant properties of coumarin. Warfarin induces its anticoagulant effect by inhibiting vitamin K epoxidereductase, which in turn diminishes production of the reduced form of vitamin K. The depletion of reduced vitamin K diminishes the γ-carboxylation of vitamin K-dependent coagulant proteins (prothrombin and factors VII, IX, and X) and the anticoagulant proteins C and S, which in turn decreases their biologic activity. Warfarin sodium is the most commonly used of the coumarin compounds in North America.[2] In addition to its use in human medicine, warfarin is a widely used rodenticide, readily available in many commercial preparations.

Warfarin has been used in attempted homicides,[3] ingested surreptitiously,[4] and administered surreptitiously by parents to children.[5,6] One of the saddest epidemics of warfarin poisoning occurred in Ho Chi Minh City (Saigon), Vietnam, in August 1981 when 177 infants died of exposure to warfarin that was transdermally absorbed through contaminated talcum powder.[7] This epidemic took place in several provinces in Vietnam and involved several different brands of talcum powder. The cause of the warfarin contamination was not discovered.

The point of contamination of the hibiscus tea in our study is not known precisely, but it is surmised to have taken place at or before the point of manufacture. Warfarin might conceivably be present in warehouses or agricultural settings. Moreover, the tea was purchased in a sealed box without evidence of tampering, and the tea came in sealed tea bags. This patient and her family's primary care physicians discount surreptitious ingestion as a plausible explanation.

To prevent the possibility of similar episodes, the patient and several of her primary care providers held a

*The international normalized ratio is a standardized method of reporting prothrombin times to ensure that a uniform intensity of oral anticoagulant therapy is used worldwide.

press conference to alert the populace to the possibility of warfarin intoxication from this product. In addition to widespread press coverage, several major television stations in San Diego reported this story. To our knowledge, there have been no other cases of warfarin intoxication from this product in San Diego.

Despite the large number of US travelers to Mexico, we think that the risk of this type of poisoning is relatively low. Nonetheless, we suggest that travelers be counseled that standards of food safety in third world countries may be considerably lower than in the United States.

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